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ANALGESIA AND ANÆSTHESIA IN OBSTETRICS: PENTOTHAL SODIUM, CYCLOPROPANE AND VINYL ETHER.*

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IT is an old problem, that of the relief of pain in obstetrics, and its solutions are still a subject of great controversy, which has been stimulated lately by the increasing demands of our women excited by the palpably specious assurances of some physicians and the active interests of drug houses. However, we must not cease to seek suitable measures of assuaging the suffering, that is, without harm to mother or child. Indeed, it is our bounden duty so to do. Let us abide by the common maxim; with end and aim to make abstruse things simple.

As a composite of experiences it is culled that of the many drugs which have been used in obstetrics for their analgesic and anæsthetic effects, almost any of them may be allowed from dependable hands, but for more universal employment we must try to find those drugs which are least injurious.

Variable degrees of analgesia, amnesia or hypnosis may occur from the use of such substances as morphine, scopolamine, paraldehyde, or the barbiturates; and also from

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the inhalation of the gaseous or volatile anæsthetics, in low concentrations. The advantages are well known, but at the same time we are fully aware of the unfavourable conditions which may ensue. I shall not discuss relative merits too exhaustively but confine my remarks more particularly to one of the newer barbiturates, namely pentothal; and to the newer anæsthetics, cyclopropane and vinyl ether (vinethene).

Great expectations were engendered from the work of Irving, Berman and Nelson[1] setting forth the benefits of nembutal. These have not been fulfilled. On the contrary there are many affirmations that even in combination with scopolamine, nembutal may cause such excitability as to necessitate the caging-in of the patient and the employment of a constant attendant. The one is not practical for ward cases, and the other is not conducive to good obstetrics. What is more, frequently, uterine contractions become definitely weakened, labour is prolonged, and the use of forceps is made indispensable. Again, often the infant is depressed to such an extent that extreme resuscitative measures are essential.

Pratt, Tatum, Hathaway and Waters[2] have found that pentothal causes *no wild excitement*, depresses respiration only in anæsthetic doses, does not disturb the circulation, and that, as this drug is broken down rapidly in the body, it closely resembles the so-called controllable anæsthetics. Accepting their report, McPhail, Gray and I[3] proceeded to use this thio-barbiturate in the early stages of labour. From 116 cases we find that there are no noticeable toxic effects on mother or foetus; that the maternal heart-rate is invariably slowed, seemingly due to the lessened excitability; and that when the foetal heart-rate is within normal limits, it is not changed by pentothal, but should it be unusually rapid, it becomes somewhat reduced by this drug. All of the infants of this series breathed spontaneously. With respect to the influence of pentothal on uterine contractions, markedly the duration of labour is shortened, an average of 9 hours for primiparæ and of 4½ hours for multiparæ. Concerning excitement, the woman is quiet, co-operative, easy to examine, and does not need special attention. We

believe that a woman does not really want to forget all about her childbirth and that from a psychical point of view it is better for her to remember something of her travail. She is no hedonist. As the drug under consideration does not produce a very pronounced degree of amnesia, we have latterly caused this to be enhanced by the administration of a small dose of scopolamine (gr. 1/150 to 1/100) at the time of the first portion of pentothal. So soon as labour pains are definitely established 4 grains of pentothal in capsule are given by mouth. One half-hour later 3 grains more are allowed and this amount is repeated at the end of one hour. Usually the patient becomes quiet, sleeps in between the contractions, and does as she is told willingly. Every half to one hour 2 or 3 grains more may be given if the effects seem to be wearing off.

Perhaps the substitute sulphur in the urea part of barbiturates may remain, and former oxygen lose its post; perhaps so to settle a heated controversy.

One should take care sedulously not to overestimate the usefulness of any one drug especially among those which have for their chief action depression of the cerebral cortex as at its best this structure possesses some functions with characteristics which easily become quite protean. Just as there is considerable choice among the so-called analgesics, so too there are several anæsthetics which themselves are useful for analgesia. We have long known the benefits of nitrous oxide in this respect, how that if anoxæmia is avoided it may be given intermittently during labour, starting quite early, without harmful effects. With the advent of cyclopropane[4, 5] it was found that an abundance of oxygen may be given with this gas and anoxia is entirely out of the question. Furthermore, it has been shown that in dogs cyclopropane anæsthesia does not damage the normal liver, even after repeated administrations, nor after long periods; that it does not increase the liver damage purposely produced by chloroform, nor impede the usual recovery of the liver from chloroform poisoning, even when the cyclopropane anæsthesia is prolonged; and that it does not cause liver impairment in starved dogs even after a three-hour period of anæsthesia.[6] Again, while cyclopro-

pane was being used in obstetrics,[7] liver function tests were carried out in three normal cases and in one of eclampsia. Practically no dye was retained in the blood of the normal women after the administration of cyclopropane, and as the liver of the eclamptic woman was damaged to an extent of more than 55 per cent before delivery, the damage was not augmented by cyclopropane, there being less than 50 per cent dye retention in her blood 22 hours after anæsthesia. At this time cyclopropane was used to produce intermittent analgesia during each uterine contraction for varying lengths of time in a number of women. In each case the termination of labour was conducted under cyclopropane anæsthesia, from which recovery was devoid of untoward effect to mother or child. Griffith,[8] one of the first to use cyclopropane, is convinced of its superiority over other anæsthestics in obstetrics. Just lately he, with Morgan and Eaman,[9] have "compared 100 cases of Caesarean section under cyclopropane anæsthesia with 100 similar operations under other types of anæsthesia, principally ethylene and ether, and report very marked improvement in the post-operative course of the cyclopropane cases particularly in regard to the factors of vomiting and abdominal distension. There was no case of adynamic ileus in the cyclopropane series whereas this had previously been a serious and fairly common complication; and there was no maternal death in the cyclopropane series. The cyclopropane cases required an average of three days less hospitalization. It is presumed that less disturbance to bowel function and less uterine hæmorrhage are the principal reasons for smoother convalescence following cyclopropane anæsthesia." I believe that the salutary feature about the use of cyclopropane in obstetrics is that an excess of oxygen is given with it.

So much for a brief consideration of the gases, nitrous oxide and cyclopropane, so much for their advantages in obstetrics, but they may not be administered by other than competent individuals. Allow me to warn against self-administration of these gases,[10, 11] that is, by the woman herself from a "fool-proof" machine. While the dangers of anoxia have been known for a long time, it is of interest to note that very recently Courville,[12] of the Department of

Neurology, Los Angeles County General Hospital, in a monograph on "asphyxia as a consequence of nitrous oxide anæsthesia," has given an exhaustive account of his studies of the effects of this form of anæsthesia on the brain in a series of 13 cases, 9 of which terminated fatally. In these a critical examination of the cerebral tissues was made. Regardless of the circumstances, the clinical symptoms and the pathological findings are the effect of *asphyxia* and are not due to any toxic effect of the nitrous oxide itself. He gives a detailed account of the damage done to the cortical cells and points out that much of this is permanent. It is easy to see that there will be a considerable number of instances wherein severe disturbances may take place without fatality. Whereas asphyxia is quite out of the question in cyclopropane anæsthesia, yet another equally serious consideration presents itself here, namely, that cyclopropane is an extremely potent drug producing anæsthesia so rapidly that it is very difficult to recognise the stages of this condition. Perhaps enough has been said to show that although these two gases occupy positions of priority in obstetrical analgesia and anæsthesia when properly used, yet their employment will not become universal.

It seems fair to say that at long last we have a substance which is safe for common use. This substance is vinyl ether (called vinethene). That it possesses anæsthetic properties was first demonstrated by Leake and Chen[13] in 1930. Later Leake, Knoefel and Guedel[14] found vinyl ether to be preferable to chloroform and ethyl ether. They declared that dogs are more easily anæsthetised, that recovery takes place more quickly, that nausea and vomiting are less marked, and that there are no significant pathological effects on the various organs. Then Goldschmidt, Ravdin, Lucké, Muller, Johnston and Ruigh[15] showed, among other things, liver necrosis in some dogs after prolonged vinyl ether anæsthesia, but no liver necrosis in monkeys, as well as no untoward effects on respiration, the circulation, the liver or the kidneys after several hundred operations of a very varied nature on patients of all ages and conditions. It was then that it occurred to me that this drug might be appropriate for anæsthesia in obstetrics, accordingly it was applied in this

capacity and the facts[16] of the first administrations of vinyl ether to parturient women show that it is easy to give and practically harmless. Since that time additional studies have been made and reported,[17, 18, 19, 20] so that now with confidence vinyl ether may be said to be "particularly suitable for obstetrical analgesia and anæsthesia in general practice on account of its safety for mother and child, its ease of administration, the rapidity of its action, the satisfactory maintenance of any desired degree of narcosis, and the early uneventful recovery".

The additional studies, mentioned above, included those on the liver,[17] from which it was found that vinyl ether anæsthesia in normal dogs does not alter the liver function appreciably; that it does not enhance the liver function damage produced previously by the inhalation of chloroform, nor does it delay the period of recovery from this damage; and that its effects upon the liver function in partially starved dogs is not appreciably different from that produced in normal animals. These findings have been confirmed separately by Molitor,[21] and by Goldschmidt, Ravdin and Lucké,[22] each of the two sets of research being conducted differently from one another and from ours. Additional studies[18] gave, as well, a most striking comparison between the liver function test findings following vinyl ether anæsthesia in parturient women and those after chloroform administered under identical circumstances. There was dye retention in the chloroform cases, indicating definite liver damage, while the administration of vinyl ether was followed by very slight dye retention and consequently practically no liver impairment.

From experiments on lower animals Molitor has shown that mixtures of vinyl and ethyl ether may have certain advantages, accordingly we[20] were led to their employment in obstetrics and can recommend the mixture of 25 per cent vinyl ether with 75 per cent ethyl ether to be given by the "open" method. In this manner analgesia is attained, at least from a practical prospect, just as quickly as it is with vinyl ether alone and the usual disadvantages of ethyl ether induction of anæsthesia are abrogated. This solves the problem of tremendous waste from rapid volatilisation which

occurs when vinyl ether alone is used by the "open" method. Hence the utility of an *improved ether* in obstetrics for the general practitioner, made easy of application, relatively inexpensive, and quite safe, that is, in comparison with chloroform or ethyl ether itself. Although we have given this mixture through a simplified apparatus (made specially for vinyl ether by the Foregger Company of New York) which consists of a closed system, fitted for supplying oxygen and for absorbing carbon dioxide, it is hardly necessary to use vinyl ether, as part of a mixture, in this fashion for the simple reason that with a "closed" machine one is not concerned with the question of volatility. Notwithstanding all of this, the fact still remains that it is infinitely better to administer this drug with oxygen. One cannot emphasise too much the advantage of giving oxygen with anaesthetics especially in obstetrics.

In conclusion, I would, at the present time, recommend pentothal sodium for the early part of labour; then, from competent hands, nitrous oxide and oxygen for intermittent analgesia should the pains become severe, and cyclopropane anæsthesia for the final stages. For those who are less experienced and for the general practitioner, vinyl ether is highly recommended to produce analgesia and anæsthesia following the use of pentothal.

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WHAT IS PHYSICAL PAIN?

FROM THE FRENCH OF RENÉ LERICHE.

(By courtesy of Anesthésie et Analgésie).

WHAT is pain of itself and without the contingencies of illness, therapeutics or medical care of any kind? Before answering that question I should like to discuss certain ideas which establish the secret unity of subjects apparently very diverse. My first theme is that the surgeon who wishes to study pain in order to suppress it must pay much attention to the complaints of his patients. He must not too lightly assume that the sufferer always exaggerates, but should listen carefully to the tale, always long and often tedious, which the patient pours out to him. No doubt almost always the patient seems to us to dwell too complacently on the complicated details of his subjective sensations, and no doubt the true doctor, by his education, must seek the facts straight away without hindrance from subjective trifles, the anatomic and pathological facts which alone can deliver up to him the secret of his patient's illness.

In the conflict which results from two opposite tendencies, that of the patient to talk about himself and that of the doctor to be objective, it is often the first which is right, and we should above all, when he is suffering, give the patient more time than we are in the habit of doing. I do not ignore the anatomical reality of the majority of illnesses, but I am convinced that there is much to be learnt by listening to patients, and I know from experience that this humane duty as a prelude to objective examination always brings real increase to our knowledge, for it gives us a better understanding of the sick man and of his sickness. There are, in fact, in illness and in the sick man more things than we are accustomed to see. It is not enough for us to identify the lesion and its cause. These are not the illness. They are but elements of it. By their

side are secondary functional disorders full of meaning. The slightest illness has more repercussions than we are wont to believe. Beyond the cause which makes, there is the temperament which permits.

We ought more and more to seek the analysis of the sick man alongside of his sickness. That discovery is not to be made in a flash. It is only by listening to the invalid, with the desire for a physiological analysis, that one can succeed in discerning the individual in his illness. Often one will find in what he says details which light up a doubtful situation which render comprehensible the mechanism of what is going on and, above all, which indicate the genesis of his pain. There remain many things to be learnt in the domain of pain and of subjective phenomena. This domain medicine has left in the air because we are taught to mistrust the subjective. This is wrong. There is here a wonderful field of study for the objective mind which with an urge for research is not content to be repaid only with words.

Another idea I would ventilate is the inadequacy of the conception of pain in illness which is put forward by the physiology of to-day. Physiological study of pain has the wrong starting point. Physiologists, following von Frey, consider pain as a normal peripheral fact, to be studied experimentally on the skin, starting with extrinsic stimuli.

This has led them to an exaggerated estimation of the problem of pain receptors. Little by little pain has come to appear as a specific sensation resulting from the activity of a sort of sixth sense. At the same time the anatomico-clinical method isolated in the spinal cord centripetal conductors of pain, discovered their selective localisation and described a pain bundle. Thus we have come naturally to a limit, a physiological entity of pain, a sensorial phenomenon existing of itself and having a warning value.

Of late the reality of this protective sense has appeared to be confirmed by the study of the approximate rapidity of specific impulses. These rapidities of conduction vary with the nature of the stimuli. Impressions of shock, of tingling, or of heat do not traverse the nerves at the same pace. Tactile impressions travel habitually at 40 metres a second,

according to Piéron, while stimuli of comparable intensity have the rates of 16 metres a second from a prick, 4½ from a burn, and 12 from a pinch. Bourginquon's time analyses gave similar precise results. The same differences have been discovered by analysing the currents of action set up in nerves. These differ one from the other as do runners going at different paces. Erlanger attributes distinct and characteristic waves to the conduction of tactile, thermal, and painful stimuli. Further, Adrian, also a specialist in these difficult inquiries, has recently come to the conclusion that there are multiple systems of pain reception, having amongst them real differences of behaviour in the presence of currents of action. Thus it appears that pain has specific receptors, special conductors of a particular diameter, along which the excitement passes at a pace which is determined for every type of pain. Faced with facts of this precision we have to admit pain as a fact of sensation, a physiological entity in itself, and no longer as an affective form of contact.

Nevertheless, my mind is not made up. I do not reject any of the experimental facts on which the theory is based. To do so would be fatuous. But I do not see their agreement with what we observe in the pain of illness. And I ask if perhaps we have not gone too far in our conclusion, beyond in fact what the experimental facts justify. I do not know if it is certain that in all these researches it is really pain that has been studied, true pain, the pain of the individual who suffers. The more I reflect upon it the less do I believe that experimental pain and the pain of illness are identical. The pain the man suffers has diverse peculiarities which differentiate it from the pain of experiment. It is not an intermittent phenomenon, the result of a brief stimulus which is repeated. It is a continuous phenomenon; it has paroxysms, but grafted on a foundation which keeps its identity through months or years.

Take this case of a man who for 18 years suffered from causalgia. During those 18 years pain never left him in peace. He slept but a few hours each night, and only with hypnotics. Waking he found his pain once more. In vain he sought to prolong that semi-consciousness which is on the threshold of re-awakening in the hope that the pain

would not come back, but it was already there, imposing itself on him and no longer to be escaped. I know well that nerves are indefatiguable. But all the same when physiologists say this they are thinking of experimental conditions. They mean something very different from this singular resistance to fatigue of a nerve which ceaselessly transmits the same pain for years. Reflect on this unlikely anomaly, which among vital phenomena would be represented by a functional activity going on permanently, varying only in the sense of a growing intensity. That is pain. Is that to be compared with the fugitive disagreeable sensation of a prick or a pinch imposed on a healthy being?

Moreover, sickness-pain does not seem to obey the ordinary laws of nerve stimulation. It has no threshold for the individual. It does not vary in intensity in accordance with the duration of the stimulus. It does not follow the law of all or nothing. I am aware that in using these words I enter a dangerous territory. I know little, to my regret, of this admirable electro-physiology which has revolutionised of recent years our ideas of nervous function. I admit everything that has been seen, but I ask that, before concluding that the pathological pain of man is a physiological and sensory phenomenon, it be studied in the person of those who suffer, those whose daily life is a contradiction to physiological law.

It is held to-day that nerve is an electrolytic conductor, the excitement of which is caused by polarisation of the ionic concentration, and that the impulse is always propagated in conditions identical for the nerve. Before admitting pain as a sensorial fact I would like to know how, from this point of view, the nerve behaves which conducts throughout years a sickness-pain.

I do not deny that the painful stimulus of the laboratory is a specific sensation. that it is conducted on the specific conditions formulated by experience. I accept the fact, since it has been demonstrated to me, that here is a sensorial equivalent. Facts graphically recorded have nothing to do with sentiment or with the individual. They are or they are not. If they are true nothing can cause them to be ignored. But I ask for the proof that we are dealing with things

identical with those others in the pain of illness, in the pain of facial neuralgia, in the causalgia of amputated limbs which lasts ceaselessly for years, in so many other painful phenomena the constancy and duration of which disconcert me. Think of the expenditure of energy represented by a facial neuralgia after thirty years of suffering or by the lightning pains of a tabetic. What an unheard-of squandering of forces. I cannot place this on the plan of the physiological facts. I may be wrong, but it seems to me there is no identity between what takes place in these instances and what one sees in the laboratory.

There is no difficulty in admitting that the modality of experimental stimulation which assumes an affective character at the centre is comparable with that which gives rise to a non-painful sensation. This stimulus travels along fibres of special diameter, with special rate; it is a mere matter of degree. But is this pain? Is this to be compared with the sensation that persists, that is renewed without cease, which is never exhausted, which knows no threshold, and the intensity of which is augmented by all kinds of intrinsic factors, which wears the individual down through months, years, and decades?

No doubt pathology is always deviated physiology. But if it is true that the laws of nerve conduction are one, can it be said that in the pain of illness there is simply the monstrous exercise of a normal phenomenon? I do not think so. It is possible that the rate of propagation of pain is in sickness different from that of touch. It is possible that the oncoming stimulus giving rise to pain produces currents of action peculiar to itself. But does this prove that in the pain of a stump there is simply the deviated activity of a normal function? The facts which we clinicians recognise as pain should be studied by the physiologists that they may see if the conclusions derived from their studies are really applicable to the object of ours. If they show that it is so I shall accept the decision. But till we have further information I find their conception too mechanical, too purely architectural, to be adapted to what we see, we the doctors of the sick man.

Again, there is this which they do not take into account,

who place so much reliance on the system of pain receptors, that almost always the pain of illness is started outside the receptor system. In trigeminal neuralgia the generating stimulus of the pain awakes in the Gasserian ganglion. In the lightning pains of tabes it is produced at the level of the posterior root or of the spinal ganglion. It seems also to have a ganglionic origin in the painful syndrome following herpes Zoster. The stimulus is produced generally far from the so-called specialised receptors.

It is only in pain of sympathetic origin, in pain set up by vasomotor, troubles, ischæmic or congestive, that the stimulus appears to arise at the level of the sensory endings of the skin, and then by a very indirect mechanism which does not in the least resemble that which takes part in experimental pain. Doubtless in the nerve conduction is indifferent. Whatever the point excited, the wave which is set up travels as though it had been produced at the very beginning of the system. But what a strange thing it would be if Nature had given us thousands of receptors for the perception of pain when almost every pain originates from outside them. And when we say nature, that means the slow acquisition of the species.

No sensation functions thus. We do not see impressions of taste, or of smell, arise spontaneously outside the stimulation of the papillæ of the tongue, or of the olfactory vesicles. We know no tactile impressions set up without the skin. When a nerve is compressed, or bears a scar, when it is divided, we get hypæsthesia or anæsthesia. Sometimes there is pain. There is never a precise sensation of touch, an hallucination of contact, precise as is the sensation of pain, which, nevertheless, we are told, originates in the same fashion.

These facts seem to me to be full of meaning. There are, we are told, specific receptors for touch, and for each of its diverse modes, for heat and cold, and for pain. The collection of these receptors assures the normal functioning of a sensation, touch. The centripetal fibres leading to the centres, specific imrpessions thus received travel side by side, each one specific in its conduction. They travel to the centres and remain physiologically distinct in the nerve.

And if one touches the nerve at any point whatever, if one pricks it from without or from within or in the middle, one never produces anything but a violent sensation of pain. One never arouses a sensation of touch or of cold. It is as though the prick never met any fibres but those of pain. When the nerve is compressed the same thing happens; sensation of pain, never sensation of touch. If the nerve is divided—outside the case of amputation when hallucination of the absent limb is brought about by a mechanism which we have explained—there is no longer sensation of touch, nor of cold, nor of heat, but painful sensations arise spontaneously, and when scarring of a nerve takes place these things persist often permanently. When a nerve is caught in a scar the wounded man experiences various painful peripheral sensations, he does not complain of normal tactile sensations. The interruption of a nerve is characterised by complete paralysis, by rapid and progressive disappearance of muscular tone, complete anæsthesia, and absence of tingling from pressure above the lesion.

When the nerve regenerates, sensations of tingling and painful, disagreeable sensations become apparent. Painful sensations always precede those of touch or of heat and cold. Everything happens as though the fibres of pain were the first to be renewed, which would truly be most singular. These painful impressions of regeneration are born on the trunk of the nerve itself, or on its branches, without there being there any receptors to receive them. When there is a skin scar the same thing happens; the scar is insensible for a long time, but it can be precociously painful, and usually it is sensation of pain which is the first to appear. In skin grafts equally it is sensation of pain which is the first to be shown or which is always the most precise. Well, neither in scars nor in grafts are there specific receptors. Then why should they exist, since they are by no means necessary for the production of sensation of pain? I repeat the physiological conception of pain is too mechanical, too coldly architectural for the subtlety of illness-pain. It ignores too much life and its shades. Some of the facts which I have related lead us to find that conception insufficient for an understanding of pathological conditions.

I have sought to show that there is pain of sympathetic origin, that is to say, a sensation the stimulation of which is received by centripetal terminations of the sympathetic system which conduct it to the ganglionic centres from where it is reflected to the spinal cord to reach the centres by the usual routes.

Knowledge of these facts seems to me to have considerable importance. It seems to me that one finds the vaso-constriction factor at the origin of a great number of painful phenomena of unknown ætiology, notably pain from cold and from accidental compression. At any rate, the results of novocain infiltrations of the sympathetic are in favour of this interpretation of the mechanism of many pains. These infiltrations, with vaso-dilating aim, are efficacious for a time in enteritis, and, generally speaking, whenever there is a permanent anatomical cause of the pain. They can assure permanent cure in those conditions when disturbed vaso-constrictor function is at the back of the pain. The interest of this method does not rest solely on the fact that it has relieved numbers of individuals, but still more that beyond its practical value it brings us a new physiological lesson. It shows us that the pain carried by a spinal nerve can be suppressed by an effect in the vaso-motor system. It makes us see the interaction of one system on the other and proves that the trouble of a function can be corrected by a purely functional effect.

This pain is not the same thing as Head's protopathic sensibility. It can be produced by direct stimulation of ganglions. Ganglions must be, in fact, an extra-rachidian marrow, "equivalent," as Morat said, "in its properties to the grey matter of the spinal cord." Erlanger and Gasser have shown that these sympathetic fibres have their own currents of action, very slow and narrow, resembling in this certain peripheral fibres. Are they too, then, conductors which produce a sensation distinct from touch, because touch does not exist at the visceral level? A theory proves its truth by being applied to all ascertainable facts. Who will dare to say that pain is a sensory phenomenon when it originates at the viscera?

Outside all conflict of ideas let us examine more facts.

Alongside of pains produced directly by pathological stimulation of centrifugal conductors or of their endings, we have seen that there is pain set up by vaso-motor disturbance—pain from vaso-dilation as in causalgia, pain from vaso-constriction as in arteritis.

This is a fact hitherto ignored which shows us the insufficiency of the present conception of the mechansim of pain in the human being. In the genesis of pain there is something more than receptors, conductors, centres and stimuli. There is something more than an anatomical construction apart from life. In the system of peripheral innervation, and doubtless also in central innervation, there is ceaseless interference by vital phenomena which by their changes and their disorders produce certain pains and paroxysms. These vital phenomena each individual fashions in his own way. This leads us to disengage the notion of the individual in the physical pain of man, the individual being the very stamp of life. It is clear that faced with a phenomenon which can produce pain we do not all react in the same way. Each of us at a stimulus is dominated by his own temperament which is interposed between the reality of the fact and the tonality of the sensation.

It is temperament which determines for each of us the very threshold of pain. One responds to the stimulus by violent pain because his sympathetic is hypertonic, because vaso-constriction is always alert in him, because his physiological being is on the edge of the pathological, and thus there arises in him for a trifle a relative ischæmia of nerve or conversely a vaso-dilation. His neighbour will have merely a passing inconvenience, a sudden disagreeable impression gone almost as soon at it arises. Other physiological phenomena play their part no doubt as well, in particular hormone values, different for each one of us. Life is not a jig-saw puzzle of functions which lie side by side and are fitted into one another. Everything in life is exchange and inter-relation. Physical pain is not a simple fact of nervous impulse travelling over a nerve at predetermined gait; it is the resultant of the conflict between a stimulus and the whole individual.

In this domain, as in the whole of pathology, it is finally,

up to a point, the humoral and vaso-motor personality which settles the behaviour of the phenomenon. This personality intervenes constantly because our organs are never other than parts of a system which are arranged for the accomplishment of a complex function. It is only by a treacherous didactic artifice that we isolate them. Thus, setting schedule aside, we ought to understand that the sensitive nerve, in spite of its appearance of morphological unity, is certainly not a physiological unity. Anatomically we know that it is the element of a complex chain which goes from periphery to centre or vice-versa. Physiologically it is but one branch in the system which assures the play of the elementary reflexes which sustain the harmony of life.

The decrees of peripheral sensibility, the reception apparatus for stimuli, the centripetal routes serve just as much to assure the obscure play of the incessant alterations of balance in the vegetative life as they do that of our movements, our postures, our acts, all matters which are strictly involved with the movements of the interior being, and it is doubtless for this reason that the life of the nerve is so narrowly blended with the life of the vegetative system. It is more so than we have ever thought. The exercise of a sensation comes only in the second place of the functional hierarchy of the nerve. The obvious function must not lead us to undervalue the basic work.

When we look at the subject thus it becomes impossible to imagine spontaneous pain, the pain of illness, as the expression of a normal activity, as the result of a sensation always awake. Pain has its place only in the order of reflex cycles. We cannot admit that it is a necessity of one's life from which there is no escape with specific apparatus and predetermined routes.

In short, the object of our analyses is not the same as that of the physiologists'. Experimental pain is for us but the fiction of a movement. With its specific receptors, its special routes, it is only a particular effective mode of touch. The pain of illness is quite a different thing; it has no receptors, no specific decrees, it cannot have, because it is not included in the normal plan of life. Such pain is

in us a kind of accident which is evolved against the grain of the laws of normal sensation. It arises usually outside the sphere of the sensory receptors, on the conductors, or in the centres; it is never exhausted; it does not weary, it increases rather than is used up by the lapse of time. Everything about it is abnormal, contrary to law. What is it in itself? It is an individual monstrous phenomenon and not a law of the species, a fact of illness. It is a disorder, the consequences of which do not allow us to view it with unconcern, which we must seek to know better in order to fight it the better. That is the aim of the surgery of to-day faced with the problem of physical pain.

THE COMBINATION OF INTRAVENOUS WITH SPINAL ANÆSTHESIA, USING PENTOTHAL AND PERCAINE

By RONALD JARMAN, D.S.C., M.R.C.S., L.R.C.P., D.A.

THE question has often been asked whether a patient should or should not be conscious of having a needle inserted into the spinal column.

The neurological physician merely wants, as a rule, to examine the spinal fluid or to insert a certain amount of liquid in order to find out the condition of the spinal trunk. He uses a little local anæsthesia and pushes the spinal needle into whichever inter-vertebral space he thinks fit. Most anæsthetists inject one of the numerous stock solutions—e.g. stovaine, planocaine, novocaine, or percaine—without any more elaborate preparation.

If the spinal injection is high, as for a high abdominal or chest operation, it may produce respiratory disturbance or a certain amount of involuntary retching of which the patient is conscious. If the patient is asleep under the effects of pentothal or nitrous oxide and oxygen, this does not—or rather, should not—happen.

During the past five years I have asked all patients who have been anæsthetised by means of percaine, whether they would prefer to be put to sleep in their beds and rendered entirely unconscious of a needle being put into the back, or whether they would prefer to be half asleep under the effects of omnopon and scopolamine and be partly conscious of a needle being inserted into the spinal column in an irksome position.

It is interesting to try to decide if one would oneself desire to be put to sleep before any type of operation, or before any type of general anæsthetic or spinal anæsthetic. and I have inquired also the personal opinion of the surgeons with whom I have worked. The answer has always

been that the surgeon would prefer to be put to sleep in his bed, and that his patients should receive the same treatment, and that, no matter what type of anæsthetic is used after induction, the patient should be entirely unconscious of the procedure.

This brings up the old controversy of whether spinal anæsthetics should be given at all, and if so, how. My answer is that it is generally recognised now, and reports have been received from all over Europe (notably from Scandinavia) and from different parts of America that spinal anæsthesia has a very definite place in the armamentarium of the anæsthetist as well as of the surgeon. It gives the maximum relaxation with the minimum shock and there is no need to soak the patient in chloroform or ether. In fact, one hardly likes to mention the word "chloroform", for as far as one can see it is fast disappearing, though the use of ether is taking a longer time to die.

The only drawbacks to spinal anæsthetics—this is a tentative statement—are the sudden collapse produced by the novocaine group and the possibility of paralysis; and the still greater bogey of headaches and backaches.

In the very large series of cases that have been given the combination of the intravenous drug pentothal with percaine, a striking point has been that the headaches formerly complained of have entirely disappeared, nor has there been any complaint of a backache; whereas when percaine was given with omnopon and scopolamine only, supplemented by nitrous oxide and oxygen, there was a definite percentage of headaches—just over 4 per cent. The combination of pentothal and percaine is given to adult patients for all major abdominal operations and may be given for operations on the hip or knee.

Now that the patient goes to sleep in his bed or actually in the anæsthetic room, according to the condition of the list in hospital, he is entirely oblivious of that fact that his back is pricked at all. Consequently he will never know that he has had a spinal anæsthetic unless he asks, and even if he is told before the operation what type of anæsthetic he will be given, he will not remember afterwards. Therefore, when he returns to consciousness he has no taste of

ether in his mouth and all that he complains of is a dryness which can be rectified by one of numerous mouth washes. There is no unpleasant smell of inhalation anæsthetic in his room or ward, and his waking can with truth be described as "coming out of a refreshing sleep".

Technique.

The technique is as follows. One hour before the operation the patient is given gr. 1/3 omnopon with gr. 1/150 scopolamine. His room is darkened or his eyes are lightly bandaged with two pads of soft wool. Then with 5 c.c. of a 10 per cent solution of pentothal, or preferably 10 c.c. of a 5 per cent solution, which does not produce a sore arm if the vein is missed, the patient is put to sleep in his bed. His mouth is held open by a Langton Hewer mouthpiece; the nurse or sister in charge supports the jaw and the patient is moved from his bed on to the trolley entirely unconscious and taken to the theatre. He is there removed from the trolley and put on to the table, and the anæsthetist has time to make himself surgically clean while the sister removes the binder and, assisted either by another nurse or the porter, sits the patient up, still supporting his jaw, and bends his back slightly forward. The back is then prepared with spirit, which is washed off with warm normal saline.

A bayonet needle is used to find the second or third space of the lumbar vertebræ, the iliac crest being the main surface marking. This needle pierces the second space and makes sure that it is clear for the spinal needle. The needle is then withdrawn and the spinal needle inserted. By experience in putting the spinal needle in one can actually feel the different spaces that it goes through, especially the extradural space before the theca is pierced. When the theca has been pierced the stylet is withdrawn, and a few drops of spinal fluid are allowed to drip away.

An important point at this stage is to make sure that the spinal needle goes into the thecal canal at right-angles. This ensures a steady flow of spinal fluid when the stylet is withdrawn, and the percaine fluid can be injected without any fear of the fluid going outside. A great fault is

where the thecal canal is only partly transfixed and an intermittent flow is produced, the anæsthetist forces the percaine in, and only part of it goes into the canal and the remainder outside; this produces a bad anæsthesia and the solution is blamed. Various users have drawn different diagrams showing how this is done, and it is desired that great stress should be laid on this point, and it should be impressed on all future users of the spinal technique.

The spinal needle being in position, 10, 12 or 14 c.c. of 1:1500 percaine heated to just over the body temperature are pushed into the thecal canal, 20 seconds and no less being taken to empty the syringe. The needle and syringe are at once withdrawn and the patient placed bolt upright. The nurses or porters doing this keep an eye on a stop-watch or a large clock with a second hand, and keep the patient upright for a time corresponding to the level of anæsthesia desired.

Etherington Wilson, who used various mock spinal columns in every imaginable position, found that a 1:1500 percaine solution rises to the level of the pubes in 25 seconds, to the level of the umbilicus in under 35 seconds, and as high as the epigastrium in under 45 seconds. Therefore, if anæsthesia of the abdomen is desired, the patient has to remain upright from the time the injection is started to the time it is finished—i.e. 20 seconds—and another 45 seconds, a total of 65 seconds. This can be exceeded up to 80 seconds in chest surgery, but beyond this it is better not to go, though there is probably a considerable margin of safety even after 80 seconds.

As soon as the proper time has expired one must work as to a stop-watch. The patient is put in the recumbent position and the table tilted to 15 degrees. The surgeon may start at once; no time is wasted and the total time, from taking the patient from his bed to the completion of the spinal anæsthesia should not exceed 10 minutes.

Conclusion.

It is desired that this article should help patients as much as the anæsthetist, as it is well recognised that the

majority of users prefer that their patients should remain conscious during a spinal anæsthetic.

The experience of the writer has been considerable, he having used percaine now ever since its inception in 1926. Several thousands of cases have been done and various methods have been used to reduce the psychic shock and to help the patient from every point of view. This method at the moment is considered to be as good as any and, according to the results, is excellent.

NOTES OF TWO CASES OF ETHER CONVULSIONS.

By R. P. W. SHACKLETON, B.A., M.R.C.S., L.R.C.P.

Case 1. *Christmas Eve*, 1932.

B.S., aged 8 years. Mastoidectomy. He had acute fulminating infection, giving a history of only 12 hours illness. Temperature 102°, pulse 120, before operation. Atropin gr. 1/120 given 15 minutes before the commencement of induction, which was carried out with open ethyl chloride on a mask, followed by ether, and was easy. Phillips's airway inserted and the anæsthetic continued with oxygen bubbled through ether and delivered into the airway. Duration of the operation, 45 minutes. A considerable amount of destruction of the bone despite the short history, with pus from the middle ear. The anæsthetic was maintained without anxiety, the depth of anæsthesia being gradually reduced. When the first skin stitch was being inserted twitching commenced in the left face and became general in about half a minute. Ether was discontinued and pure oxygen given. Head raised. The convulsions continued with a series of clonic spasms followed by short remissions. After 15 minutes calcium glucinate (Sandoz), 10 c.c. of a 10 per cent solution was given intravenously. The convulsions ceased suddenly shortly after this. Oxygen was given throughout. The theatre was warm but not unduly hot. It was a very cold day outside. The following day the boy was very ill. A right basal pneumonia had developed. There were signs of meningism. Lumbar puncture showed fluid under high tension and turbid. Cerebral abscess was later localised to the left temporal region.

The second operation was four days after the first. Temperature 105°, pulse 112, respiration 40. Atropine gr. 1/120 was given. Induction with C_1E_2 using ℥iv of the mixture. Subsequently ether ℥jss in all over 40 minutes.

At the end of this operation the boy's condition was fair, pulse 120. No sign of any convulsions. Theatre temperature about the same as previously. Despite all treatment the boy died 12 hours after this operation.

Points in Case 1 which might receive attention.

1. Was it a true ether convulsion in view of the cerebral symptoms demonstrable the next day? (Anyway, what is a true ether convulsion?)

2. The very toxic state of the boy. (Much more toxic at the time of the second operation when no convulsions occurred. Certainly much less ether was used for this anæsthetic.)

3. The convulsions only occurred at the end of the operation (45 minutes), when the anæsthetic was comparatively light.

4. It coincided with a definite stimulus (the insertion through the skin of the first stitch).

5. The convulsions ceased about five minutes after calcium glucinate had been given intravenously.

6. The second operation four days later without any repetition.

7. The temperature of the theatre was not abnormal on either occasion. It was very cold outside.

Case 2.

Also a mastoidectomy. A boy, aged 10 years, following measles. He had had several days otitis media. Was toxic. (Temperature 101°, pulse about 100.) Both mastoids were infected and both were dealt with. Atropine gr. 1/120 given 15 minutes before operation. Induction by open ethyl chloride—ether sequence. Ether continued up to fairly deep anæsthesia when oral endotracheal intubation with a Magill's catheter was performed. A Boyle's dry bobbin flow-meter machine was used, the anæsthetic being maintained by $N_2O + O_2$ with minimal ether. (I have unfortunately no note of the amount of ether used in this case. My recollection is that it was fairly small.) The anæsthetic was uneventful until the insertion of one stitch in the second mastoid wound, the anæsthetic being

light at this moment preparatory to letting the boy out with a CO_2+O_2 period, when convulsions suddenly commenced. The sequence was as before, face, limbs, trunk. The convulsions lasted five minutes, and the sole treatment was CO_2+O_2 (the endotracheal catheter still in position), and raising the boy's head. Calcium was about to be given when the convulsions ceased. The boy's recovery was uneventful.

The points about this are are:

1. Like my first case, a mastoid.
2. The presence of toxæmia.
3. The comparative lightness of the anæsthetic when the convulsions occurred. (In each case.)
4. The stimulus of the stitch. (In each case.)

The theatre temperature was not noted, neither do I remember if it was very warm.

When these cases occurred not much attention had been paid to the subject. One was in 1932 and the second in 1935.

PRESENTATION TO LORD NUFFIELD

DURING his recent visit to the United States the Nuffield Professor of Anæsthetics, Mr. R. R. Macintosh, was asked to receive on behalf of Lord Nuffield a silver plaque presented by the International Anæsthetic Research Society and the International College of Anæsthetics. The plaque is engraved "Lord Nuffield, O.B.E., D.C.L., in appreciative recognition of his generous vision in founding and endowing the Professorship of Anæsthetics at the University of Oxford, as another milestone in the world conquest of human pain. Presented by the International Anæsthetic Society and the International College of Anæsthetists. 1937."

DIPLOMA OF ANÆSTHETICS

AT a quarterly convention of the Royal College of Physicians held on July 29th last, Mr. C. W. Morris, M.B., was elected examiner for the ensuing year. Mr. Morris thus joins Mr. H. E. G. Boyle who, as we have already notified, was appointed by the Royal College of Surgeons for the same period.

The following practitioners have been granted the Diploma in Anæsthetics:—M. C. Canguli, R. E. Pleasance, A. F. Potter, W. B. Primrose, W. Sankey, G. E. P. Meldow, H. D. Merrington, C. D. Banes, R. P. Booth, Elva M. Chivers, R. T. V. Clarke, R. W. Cope, H. Curtis, G. D. Drury, A. C. Fraser, A. H. Galley, J. L. Hopkins, G. Landau, I. N. Lewis, H. J. V. Morton, A. H. Musgrove, J. A. V. Nicoll, W. Niven, Angela M. S. A. Openheim, G. S. W. Organe, F. H. Pratt, A. C. R. Rankin, O. N. Ransford, J. R. Ritchie, Major J. M. Savege, R.A.M.C., Major H. R. Shepperd, R.A.M.C., J. T. Turner, R. C. Walch, Doris E. G. Walker.

ABSTRACTS

"Observations on the viability of leucocytes following the administration of anæsthetics." By Dr. MARIO FERNANDEZ, University of Messina. *Annali Italiana di Chirurgia*. February 1937, p. 137.

THE author has made observations on 48 patients and 18 animals. Of the former, 18 were operated on under ether anæsthesia, 6 under chloroform, 8 under spinal, and 16 under local anæsthesia. Six of the animal experiments were made under ether, 6 under chloroform, and 6 without any anæsthesia.

From the observed results the author concludes that the length of survival *in vitro* of leucocytes is almost always reduced after the administration of a general anæsthetic. In the cases anæsthetised with ether the reduction in survival time is from two to four hours. In those submitted to chloroform the reduction of life is from one to three hours. Local and spinal anæsthetics do not appear to have any effect on the viability of the leucocytes: in only one case was any marked change observed, a three-hour reduction. In the animal experiments conducted without anæsthesia, no loss of vitality was observed in the leucocytes and it therefore appears that operative trauma is not responsible for any of the effect noticed in the anæsthetised cases.

Further, it was observed that the length of time the anæsthetic was administered produced no difference in the effect on the leucoyctes; those cases which had long operation showed similar results to those in which the anæsthesia was very short. The white blood-count in all cases for which ether and chloroform was used was found to be less after the operation than before, whilst the movements of the leucocytes at first sight appeared to be more active than normal.

"Headache after spinal anæsthesia." By KOSTER, KASMEN and SHAPERO in *Archives of Surgery*, July 1937, p. 148.

The authors desired to find out by clinical experiment

whether there is any truth in the idea that contamination of the injected solution, or its hyper- or hypo-tonicity is responsible for headache after endothecal injection. They tried out various solutions on groups of 25 patients. Their results led them to infer that the addition of tincture of iodine, or of a solution of trinitrophenol, the antiseptics in use to cleanse the skin before injection, to procaine hydrochloride dissolved in cerebro-spinal fluid does not add to the risk of headache post-operatively. Further, hypertonicity or hypotonicity as occasioned by using tap water, saline solution or distilled water, is not a factor in the production of headache. The authors assume that either seepage of cerebro-spinal fluid, irritative meningitis, or contamination of injected material causing irritation of nerve tissue, is regarded as the cause of post-operative headache. Their work, so far as it goes, disproves the second supposed cause, but does not get us any nearer to knowing what the real cause is.

"Dilaudid-scopolamine before evipan anæsthesia." By A. Riès in *Schmerz Narkose-Anæsthesia*, June 1937, p. 34.

The author desired prolongation of evipan narcosis and abolition of the clonic spasms often seen in its early stages. After trying other premedication he met the greatest success by injecting, one hour before the evipan administration. 1 c.c. of a solution of dilaudid and scopolamine. This gave a dosage of 0.002 gr. dilaudid and 0.0003 scopolamine. He does not exceed 10 c.c. of evipan and usually needs not more than 8 c.c. for a perfectly satisfactory short anæsthesia with good relaxation of abdominal muscles. The duration of anæsthesia is twice as long as without premedication. Restlessness and sickness afterwards are very rarely seen. The recovery period is prolonged but it is quiet and comfortable.

"Effect of analgesia and anæsthesia in the newborn infant." S. H. Clifford in *Journal of Medicine*, February 1937, p. 612.

From the study of a number of normally delivered full-

term infants the author arrived at certain conclusions respecting the effects of sedatives and narcotics used during labour. His opinions regarding morphia are those hitherto commonly held, viz. an unfavourable influence on the baby if the drug is given too freely or too near to the time of delivery. The barbiturates he found to have no ill effect except that the infant at birth is sleepy with relaxed muscles; in 3 per cent of the cases, however, artificial resuscitation was needed. His conclusion regarding nitrous oxide is interesting, for he finds that a concentration above 85 per cent exerts a profound influence through the production of intra-uterine anoxæmia and a concentration of 90 per cent nitrous oxide with 10 per cent oxygen, for five minutes or more, will produce severe asphyxia in three out of five newborn infants.

"Anæsthetic methods and post-operative respiratory complications." By TAYLOR, BENNETT and WATERS in *Anæsth. and Analges.*, July-August 1937, p. 187.

IN a three year's statistical report from the Wisconsin University's Department of Anæsthesia the authors state some valuable conclusions. The first we note is that in bad risk patients the skill and judgment of the anæsthetist are more important than the agent used. Cyclopropane, which was largely used in various types of operation, comes out very favourably in the list of agents. Unless there is faulty technique post-operative complications cannot justly be put down solely to anæsthesia. The most important factors influencing post-operative respiratory complications were degree of narcosis, duration of operation, type of operation, and surgical risks. Most observers would be inclined to add to this list pre-existing infection of respiratory passages.

"The relation of hyperpyrexia to anæsthesia." By J. BARANGER in *Anæsth. et Analges.*, July 1937, p. 385.

THE author draws attention to the danger of certain anæsthetics when given to those who are suffering from high temperature. He was attracted to the subject by a patient, 18 years of age, who under oil ether rectal anæsthesia had

a wisdom tooth and abscess of the jaw removed. The temperature at the time was tropical. Anæsthesia and operation were apparently normal and on return to bed the patient was given a rectal wash out. After half an hour there was respiratory collapse necessitating artificial respiration for 30 minutes. Tiffenham has investigated the matter experimentally and shown that in the presence of high temperature elimination of anæsthetics from the brain matter is greatly delayed. During anæsthesia the heat-regulating centre does not function. Consequently the body heat is raised unduly by the surrounding excessive temperature and the undue storage of anæsthetic vapour occurs.

CORRESPONDENCE

Analgesia in Labour.

To the Editor.

Sir,—Dr. Minnitt has courteously drawn my attention to his note, written in conjunction with Dr. Hilda Barry, in the *Journal* of April (Vol. XIV, No. 3), which criticises my method of inducing analgesia in the parturient woman. (*Proc. Roy. Soc. of Med.*, September 1937, and *Lancet* March 13th, 1937.) The essential feature of this method is the inhalation by the patient of a limited amount of nitrous oxide gas before the onset of each "pain" (110 oz. in two or three deep breaths.) It is now suggested that the analgesia so produced must be insufficient in duration to be effective in relieving the pains of labour. As it appears to me that Dr. Minnitt is misled on certain points and as his note may also be misleading to others, I should like to state the essential facts as I see them.

Dr. Barry gives figures of the observed duration of labour pains. Some of her patients had astonishingly long pains, and I can only suppose that the figures refer to the total duration of a uterine contraction as determined by palpation, and that they include the period of uneasiness which precedes and follows each "pain". But this is irrelevant. Nitrous oxide is given with the object of relieving the distressing severe pains of labour, and these in my experience seldom endure for as long as 60 seconds and are frequently of much shorter duration. Nor are the irregular, prolonged, or rapidly succeeding pains of the late second stage under discussion, for, as I have elsewhere said, I consider that a stronger anæsthetic agent should be used at that period. Now, as I have shown, the symptoms induced by the inhalation of two or three deep breaths of pure gas continue for some 70 or 80 seconds. Thus, contrary to the inference made in Dr. Minnitt's and Dr. Barry's notes, the duration of the patient's suffering is in

the great majority of cases well covered by the effects of a single administration of gas.

The argument, however, does not stop here, for no mention is made of the fact which I have so strongly emphasised, namely, that analgesia is not instantly produced by inhalation of nitrous oxide. Pure gas takes 16 seconds to act, while a 35 per cent gas-air mixture as used by Dr. Minnitt requires as much as 30 seconds; and the effect of the continued inhalations is even then far less in magnitude than the effect of two to three deep breaths of pure gas. Now, women in labour have but short warning of the onset of each new pain; 10 to 15 seconds is a usual time. From these facts it will be realised that the period of analgesia induced by the inhalation of pure gas much more closely coincides with the period of the patient's sufferings than does the analgesia produced by a gas-air mixture. Dr. Minnitt's criticism is thus not only nullified, but is actually reversed.

More convincing than theoretical argument is the actual experience gained in the labour room. After a fair trial of both methods I have no hesitation in saying that for obstetrical purposes the inhalation of a limited quantity of pure nitrous oxide gives results which are distinctly superior to those produced by intermittent inhalations of a 35 per cent gas-air mixture. Patients who have been instructed in the use of both types of machine and who have been left to form their own judgment have almost without exception expressed a strong preference for the type which enables them to inhale pure gas before each pain.

I apologise for writing at such length, but I am impelled to do so by the thought that Dr. Minnitt's remarks may discourage the use of a method of relieving the pains of labour which, although not perfect, has nevertheless given me results which are a considerable improvement on those which I previously obtained with nitrous-oxide-air mixture.

I am, Sir, Yours faithfully,

CHASSAR MOIR,
Reader in Obstetrics and Gynaecology, British Postgraduate Medical School.

14th September, 1937.

Ether Convulsions.

To the Editor,

Sir,—The following case might be of interest to anæsthetists. A youth aged 16 was admitted to Wellhouse Hospital, suffering from osteomyelitis of the humerus, on a warm Sunday afternoon. His temperature on admission was 103 and the pulse 120. He was extremely ill, and it was decided to operate.

Gas-oxygen-ether was used for the induction and a Magill tube passed through the nose without difficulty in the anæsthetising room. The patient was then taken into the theatre and placed on the table and prepared for the operation.

About five minutes later, that is to say, some 12 minutes after the anæsthetic was started, muscular movements were noticed round the lips and eyelids, and to overcome this anæsthetic was slightly deepened, whereupon the movements became more marked, spread over the whole body and the patient went into violent convulsions. He became cyanosed, but the respiration did not cease.

The ether was turned off and pure oxygen was given through the Magill's tube, when his colour improved, but the convulsions did not cease.

Chloroform was then given, but as the anæsthetic was deepened with the chloroform the convulsions became more marked and the patient more cyanosed.

The operation was completed with the assistance of the nurses holding the patient's arm in position, and when the operation was completed the convulsions became less severe.

Carbon dioxide-oxygen was given, still through the Magill's tube, and as the patient "came round" from the anæsthetic the muscular movements decreased in severity. As the colour was good it was decided to remove the Magill's tube. Before this was done a Hewitt's airway was placed in position in the mouth, and as the patient was by now practically "round" from the anæsthetic, the tube was

withdrawn. All convulsions ceased, and the patient gave no further trouble and is now on the road to recovery.

Yours faithfully,

JOHN ELAM,
Visiting Anæsthetist, Wellhouse Hospital, Barnet;
Honorary Consulting Anæsthetist, British Hospital for Mothers and Babies, Woolwich.

July 1937.

REVIEW

"The technique of local anæsthesia." A. E. HERTZLER, A.M., M.D., etc., Professor of Surgery in the University of Kansas. Sixth edition, 284 pages, 142 illustrations. Henry Kimpton, London 1937. Price 21*s*.

DR. HERTZLER lays stress on the advisability of using minimum amounts of solution when practising local analgesia. The advice is no doubt good and entails accurate anatomical knowledge of nerve-supply and distribution if it is to be effectively carried out. Obviously it is far better if, thus equipped, the surgeon can get the analgesia he wants without an indiscriminate widespread infiltration which alters the relations of the parts and is always liable to interfere with healing or engender unwanted results from absorption. The author wisely advocates careful selection of patients for local methods; he is no narrow partisan for his favourite procedure in every case. For example, he recommends general anæsthesia for removal of tonsils in young children, but in adults generally regards this as "an unnecessary infliction on the patient." For operations on larynx and trachea he thinks local analgesia always the choice. Discussing the method for thyroid operations he makes no mention, which is a pity, of the psychical aspect of the problem in patients with exophthalmic goitre. Surely here is an instance in which the mental attitude of the patient often necessitates if not a general anæsthetic at any rate the need of preliminary sedatives or narcotics. He regards operations on the abdominal contents as belonging "distinctively to the domain of general or spinal anæsthesia," but regards splanchnic anæsthesia as holding out much hope in the future. This passage was originally written. we fancy, before Finsterer and Gordon Taylor had published their many records. The book is an excellent one from which to learn the practice of local analgesia, although, as always in this connexion, the admirable illustrations make the procedure look easier and more clear cut than ever it is in practice.

OBITUARY

WE regret to record the death of a much respected senior anæsthetist, Mr. Carter Braine, who bore a name long famous in English anæsthetics. For thirty years he was "surgeon anæsthetist" to Charing Cross Hospital, where his father, Francis Woodhouse Braine, F.R.C.S., had been "chloroformist" in the days when J. T. Clover was making anæsthetics a speciality at University College Hospital. Carter Braine was appointed to Charing Cross in 1890 and retired under the age limit in 1919. He also held appointments at the Royal Dental and St. Peter's Hospitals. A sound anæsthetist, Carter Braine taught more by example than by precept and his equable temperament endeared him both to students and to colleagues. In his later years he confined his work almost entirely to dental anæsthetics. Carter Braine leaves a widow and two sons, one of whom is assistant surgeon to the X-ray Department of Guy's Hospital.

ERRATUM

In the last number of the *Journal* (July 1937) Dr. Stanley Rowbotham was described as "Anæsthetist, King's College Hospital," etc. This should have read "Anæsthetist, Charing Cross Hospital," etc., and we apologise for the mistake.